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Preoperative Left Ventricular Function: Minimal Requirement for Successful Late Results of Valve Replacement for Aortic Regurgitation

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Postoperative survival and left ventricular function were studied in 62 patients who underwent aortic valve replacement for isolated, chronic aortic regurgitation between 1978 and 1985. The average follow-up period was 3.8 years. There were three in-hospital and six late deaths. Five (56%) of the nine postoperative deaths were of cardiac-related causes. The mean 7 year survival rate was 83 ± 5%. Preoperative left ventricular end-systolic volume index was the most important indicator ($p < 0.001$) for subsequent cardiac death. The 6.5 year survival rate was 92 ± 4% for patients with an end-systolic volume index <200 ml/m² compared with 51 ± 16% for those whose index was >200 ml/m². None of the 48 patients with an end-systolic volume index <200 ml/m² died of cardiac-related causes.

Twenty-three of the 48 patients with an end-systolic volume index <200 ml/m² (Group 1) and 6 of the 12 patients with a higher index (Group 2) underwent repeat catheterization 26 months postoperatively. Preoperative afterload, assessed by end-systolic wall stress, was elevated in both groups, but decreased postoperatively, becoming identical to the afterload in 20 normal control subjects. Although the preoperative ejection fraction was depressed in both groups, the great majority of patients in Group 1, compared with none in Group 2, exhibited normal ejection fraction postoperatively.

Thus, in patients who recently underwent surgery for aortic regurgitation, satisfactory late results in both long-term survival and reversal of left ventricular dysfunction were obtained when the preoperative end-systolic volume index was <200 ml/m².

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The timing of valve replacement in patients with aortic regurgitation constitutes a clinical dilemma and is based on postoperative survival and the degree of hemodynamic improvement after surgery. Numerous angiographic and echocardiographic studies have dealt with this subject. Several studies (1–7) have suggested that preoperative left ventricular function is an important determinant of postoperative prognosis. However, Fioretti and coworkers (8,9) and Daniel et al. (10) reported excellent postoperative survival regardless of left ventricular dysfunction. Thus, previous studies have had conflicting results, which can be attributed primarily to differences in patient selection and techniques of perioperative myocardial preservation.

In the present study, we reevaluated the results of aortic valve replacement in patients with aortic regurgitation who had undergone an operation using current myocardial protection techniques consisting of cold potassium cardioplegia. Postoperative survival and hemodynamic alteration were compared in patients with and without severe preoperative left ventricular dysfunction. Before and after operation, left ventricular wall stress and ejection performance were examined to analyze changes in contractile function of the left ventricle. The purpose of this study was to determine the minimal preoperative left ventricular function necessary for satisfactory postoperative survival and hemodynamic results in the current operative series of patients with aortic regurgitation.

Methods

Study patients. The records of all patients who underwent aortic valve replacement for isolated, chronic aortic regurgitation at Osaka University Hospital between July 1978 and June 1985 were reviewed. Patients with significant aortic stenosis (mean aortic valve gradient >20 mm Hg), other valvular lesions or coronary artery disease were ex-

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cluded. Of the 64 patients identified, 2 were excluded because of inadequate data for analysis, which left 62 patients for this study. There were 48 men and 14 women, with a mean age of 43 ± 12 years (range 18 to 64) at the time of aortic valve surgery. The origin of aortic regurgitation was rheumatic disease in 34 of these patients, remote aortic valve endocarditis in 9, congenital bicuspid valve in 7, syphilis in 3, aortitis in 4, Marfan's syndrome in 1, annuloaortic ectasia in 2 and unknown in 2. Six patients were subjectively asymptomatic, 25 were minimally symptomatic (functional class II of the New York Heart Association) and 31 were severely symptomatic (class III or IV). Fifty patients (81%) were receiving digitalis.

Indication for aortic valve replacement was based on recognition of one or more of the following: angina pectoris, presyncope or syncope, congestive heart failure and evidence of cardiac enlargement on the basis of serial chest X ray films or echocardiography. No patient was denied the operation because of severity of preoperative left ventricular dysfunction.

Surgical technique. All patients underwent aortic valve replacement with a Björk-Shiley tilting disc prosthesis within 3 months of catheterization. The average aortic-cross clamp time was 69 ± 20 minutes. Myocardial protection during the procedure was provided by cold potassium cardioplegia and topical cooling with ice slush. The dose of cardioplegic solution was determined by the left ventricular mass; the initial infusion was a solution of ≥3.0 ml/g of the left ventricular mass. We have previously reported that in 28 patients with chronic aortic valve disease and left ventricular hypertrophy, the peak postoperative creatine kinase isoenzyme MB (MB CK) levels were low in those patients with an initial volume of cardioplegic solution of ≥2.5 ml/g of left ventricular mass regardless of the size of the left ventricular mass (11).

Preoperative cardiac catheterization. Preoperatively all patients underwent right- and retrograde left heart catheterization and angiography, performed in the postabsorptive state with mild sedation (pethidine hydroxychloride, 1 mg/kg, and hydroxyzine, 1 mg/kg, both intramuscularly). Pressures were measured with a fluid-filled catheter connected to Statham P23Db or P23ID strain gauges, and tracings were recorded at a paper speed of 100 mm/s with an eight channel optical recording system (Hewlett-Packard). Aortic and left ventricular pressures were recorded immediately before left ventriculography, during which time there was no change in heart rate. In eight patients, micromanometer-tipped angiographic catheters were used, permitting high fidelity recording of left ventricular pressure simultaneously with left ventricular cineangiography. Cardiac output was determined by the dye-dilution method. Aortic root angiography was performed on all patients to estimate the degree of aortic regurgitation. Left ventriculography was carried out with biplane 35 mm cineangiography (30° right anterior and 60° left anterior oblique) in 47 patients, and single plane 35 mm cineangiography (30° right anterior oblique) in 15 patients. Left ventricular cineangiograms adequate for calculating left ventricular volume and ejection fraction were available for 60 of the 62 patients. All patients >40 years of age, as well as those experiencing chest pain on exertion and those suspected of having coronary artery disease, underwent coronary arteriography.

Left ventricular measurements. Left ventricular volumes were determined from the left ventricular cineangiogram using the area-length method, in conjunction with a small computer interfaced with a digitizer. Correction factors for X-ray magnification and pincushion distortion were obtained with a 1 cm^2 calibrated grid system. Special efforts were made to ensure that the position of the grid in space and its relative distance from the image intensifier was identical to that of the patient's left ventricle. Volumes were calculated using the regression equation for the right anterior oblique position derived by Wynne et al. (12). The earliest well opacified cardiac cycle was selected for analysis, with the exclusion of extrasystolic and postextrasystolic beats. Six patients had atrial fibrillation, their left ventricular volumes having been calculated as the average of three consecutive cardiac cycles. We found close agreement between stroke volume determined by angiography and by the dye-dilution technique in 20 normal subjects: 84.4 ± 18.2 and 80.6 ± 17.8 ml ($r = 0.88$, $p < 0.001$), respectively.

End-systolic volume (ESV) was defined as the smallest ventricular volume, and end-diastolic volume (EDV) as the largest ventricular volume from serial frames. Ejection fraction was calculated as (EDV − ESV)/EDV. The left ventricular long axis (L) and area (A) were measured in the right anterior oblique projection, and the short axis (D) was calculated as $D = 4A/\pi L$. Wall thickness was measured at the midanterior wall of the right anterior oblique ventriculogram at end-diastole. Left ventricular mass was determined at end-diastole by the method of Rackley et al. (13). End-systolic circumferential wall stress was calculated from Mirsky's formula (14) as $(Pb/h)(1 - b^2/2a^2 - h/2b + h^2/8a^2)$, where P = left ventricular systolic pressure, h = wall thickness and a and b = the midwall semimajor and semiminor axes at end-systole, respectively. The result obtained by this formula was converted to dynes × $10^3/cm^2$ by multiplying by 1,332 dynes/cm^2 per mm Hg. End-systolic wall thickness was calculated from end-systolic volume and left ventricular mass, which is assumed to be constant, according to the method of Hugenholtz et al. (15). In this study, left ventricular peak systolic pressure was substituted for end-systolic pressure. The left ventricular mass and end-systolic wall stress were not determined preoperatively in patients with pericardial effusion demonstrated by echocardiography or found at operation, or both.

Postoperative catheterization. Twenty-nine patients, 25 men and 4 women, underwent repeat cardiac catheter-

Table 1. Characteristics of Nine Patients With Aortic Regurgitation Who Died After Aortic Valve Replacement

Patient No.	Age (yr) & Sex	NYHA Class	CTR (%)	EDVI (ml/m²)	ESVI (ml/m²)	EF	Postoperative Survival (mo)	Cause of Death
1	33M	IV	75	376	291	0.20	1	CHF
2	38M	III	61	294	230	0.22	2	CHF
3	38F	III	70	281	157	0.44	4	Respiratory failure
4	40M	III	70	437	317	0.27	49	CHF
5	48M	II	62	308	203	0.34	4	Sudden
6	29M	III	68	516	351	0.32	37	Sudden
7	33M	III	62	270	151	0.44	25	Infective endocarditis
8	63M	IV	60	—	—	0.36	7	Cerebral hemorrhage
9	31M	II	56	—	—	—	2	Suicide

CHF = congestive heart failure; CTR = cardiothoracic ratio; EDVI = end-diastolic volume index; EF = ejection fraction; ESVI = end-systolic volume index; F = female; M = male; NYHA = New York Heart Association functional classification.

ization at a mean of 26 months (range 5 to 80) postoperatively after having undergone uncomplicated aortic valve replacement. The indications for repeat catheterization were not selective. The purpose of the restudy as well as the invasive nature of the tests were explained in detail to all patients. Patients who gave informed consent underwent restudy; none of them had complications at initial or repeat catheterization. Catheters were positioned transseptally in the left ventricle by means of femoral vein puncture, and retrogradely in the ascending aorta by means of femoral artery puncture. Postoperatively, aortic pressure was recorded just before and during left ventriculography. Pre- and postoperative studies were performed using the same equipment and using the same catheter manometer systems. Coronary angiography was repeated if it was clinically indicated. Peak systolic gradient across the prosthetic valve was <10 mm Hg in 24 patients, 10 to 20 mm Hg in 3 patients and >20 mm Hg in 2 patients. Two patients were experiencing atrial fibrillation during preoperative evaluation, but both exhibited sinus rhythm after the operation.

Normal subjects. For comparative purposes, normal values for quantitative angiographic and left ventricular hemodynamic data were obtained from 20 normal subjects who underwent cardiac catheterization because of an atypical chest pain syndrome.

Follow-up. Postoperative follow-up data were obtained from outpatient clinics or through personal interview with patients or their families, or both. The closing date for follow-up was December 1985. The average follow-up period was 45 ± 26 months.

Statistics. All data were expressed as mean values ± SD. Survival curves were calculated according to the method of Kaplan and Meier (16). Comparisons of preoperative and postoperative data were made using the paired *t* test. Unmatched variables were compared with the unpaired *t* test or Wilcoxon rank-sum test (17). Comparisons among continuous variables in more than two groups were made with one-way analysis of variance (ANOVA), followed by Newman-Keuls' multiple comparisons if the ANOVA probability (p) value was <0.05. The significance of variables as independent predictors of surgical outcome was determined by stepwise multiple discrimination analysis.

Results

Postoperative survival (Table 1). None of the 62 patients died within 30 days after operation, but 3 died later in the hospital. Two of the three died from congestive heart failure 31 and 61 days, respectively, after operation; the third died of progressive respiratory failure and septicemia 4 months postoperatively. Six patients died after hospital discharge. One patient died from progressive congestive heart failure 49 months after surgery and two patients died suddenly 4 months and 37 months, respectively, after surgery. In the latter patient, repeat cardiac catheterization 9 months postoperatively showed persistent severe left ventricular dysfunction, with an ejection fraction of 0.33. One patient died of prosthetic valve endocarditis, which was confirmed at autopsy. The remaining two patients died of noncardiac causes. Thus, a total of nine patients died after operation, five (56%) of the postoperative deaths being cardiac in origin. The 7 year postoperative survival rate was 83 ± 5% (Fig. 1). Excluding the four noncardiac deaths, the 7 year survival rate was 89 ± 5%.

Preoperative left ventricular function and survival (Table 2). The five patients who died postoperatively of cardiac-related causes, when compared with the late survivors, showed a significantly higher cardiothoracic ratio, left ventricular end-diastolic pressure, end-diastolic volume index, end-systolic volume index and left ventricular mass index, as well as a lower ejection fraction (Fig. 2). There were no significant differences in age, functional class, cardiac index or left ventricular systolic pressure.

Figure 3 plots the preoperative ejection fraction versus the preoperative end-systolic volume index; all five patients who died of cardiac-related causes had an end-systolic vol-

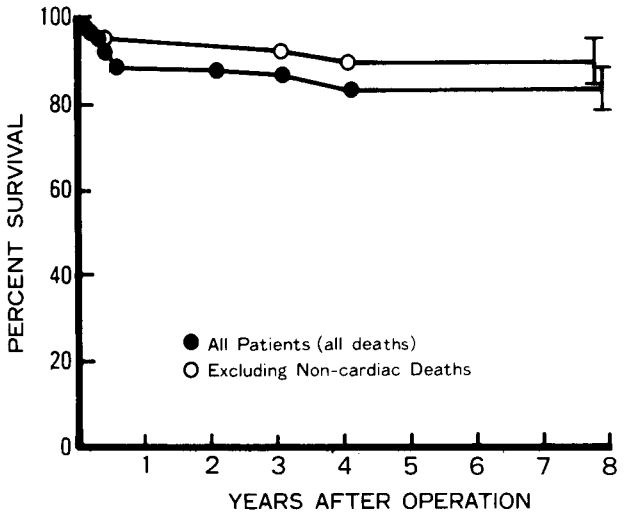


Figure 1. Postoperative survival in 62 patients with aortic regurgitation. Brackets indicate SD.

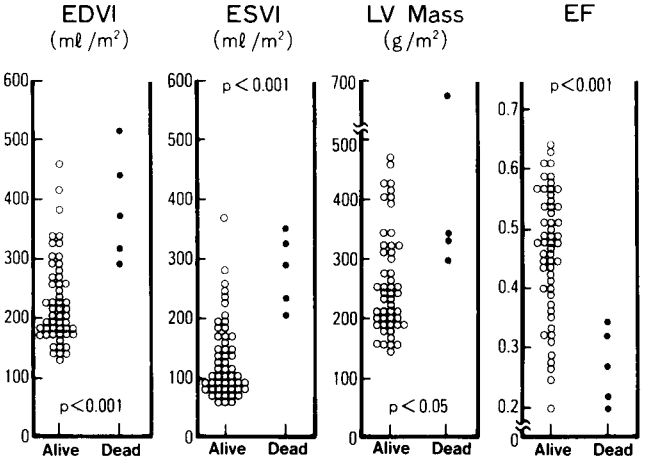


Figure 2. Individual values for preoperative variables are plotted for 53 patients surviving (Alive) after aortic valve replacement and 5 patients who died suddenly or from heart failure (Dead) after operation. EF = ejection fraction; EDVI = end-diastolic volume index; ESVI = end-systolic volume index; LV Mass = left ventricular mass index.

ume index >200 ml/m^2 and an ejection fraction <0.35 preoperatively. Five (42%) of the 12 patients with a preoperative end-systolic volume index >200 ml/m^2 died of myocardial causes and the actuarial survival rate at 6.5 years postoperatively for these 12 patients was 51 ± 16% (Fig. 4). In contrast, postoperative survival of 48 patients with a lower end-systolic volume index was excellent (no cardiac deaths).

The preoperative variables of age, functional class, cardiothoracic ratio, left ventricular end-diastolic pressure, end-diastolic and end-systolic volume indexes and ejection fraction were subjected to stepwise multiple discrimination analysis. The most important indicator for postoperative cardiac death was the preoperative end-systolic volume index ($p < 0.001$).

Postoperative left ventricular function (Tables 3 and 4). Of the 29 patients who underwent repeat catheterization postoperatively, 23 (Group 1) had an end-systolic volume index <200 ml/m^2 and 6 (Group 2) had an end-systolic volume index ≥200 ml/m^2 preoperatively. There were no significant differences between the two groups in age, interval from operation to postoperative study, size of Björk-Shiley prosthesis or peak systolic pressure gradient across the prosthetic valve. The average cardiothoracic ratio was markedly higher in Group 2 than in Group 1 patients, but there was substantial overlap between the two groups. For comparable heart rates before and after operation, the left ventricular systolic pressure did not change significantly in either group after operation, whereas the left ventricular end-diastolic pressure decreased and the cardiac index increased remarkably. Postoperatively, there were no significant differences in left ventricular end-diastolic pressure and cardiac index between normal subjects and the two patient groups.

Table 2. Preoperative Data for Patients Surviving After Aortic Valve Replacement and for Patients Who Died Suddenly or From Heart Failure After Operation

	Alive (n = 53)	Cardiac Death (n = 5)	p Value
Age (yr)	44 ± 12	38 ± 7	NS
NYHA class	2.5 ± 0.9	3.0 ± 0.7	NS
CTR (%)	59 ± 7	67 ± 6	<0.02
CI (liters/min per m^2)	2.78 ± 0.79	2.44 ± 0.88	NS
LVSP (mm Hg)	146 ± 26	123 ± 17	NS
LVEDP (mm Hg)	16 ± 8	24 ± 10	<0.05
EDVI (ml/m^2)	227 ± 80	386 ± 92	<0.001
ESVI (ml/m^2)	127 ± 66	278 ± 61	<0.001
LVMI (g/m^2)	261 ± 89 (n = 50)	403 ± 168 (n = 4)	<0.05
EF	0.46 ± 0.11	0.27 ± 0.06	<0.001

Data are means ± SD. CI = cardiac index; LVEDP = left ventricular end-diastolic pressure; LVMI = left ventricular mass index; LVSP = left ventricular systolic pressure; other abbreviations as in Table 1.

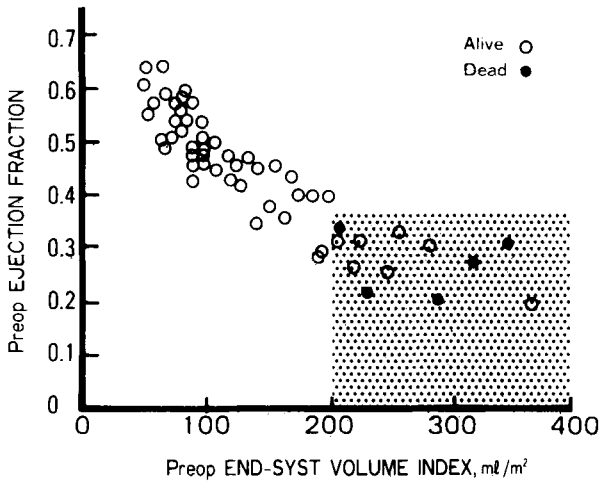


Figure 3. Plotting of preoperative (Preop) left ventricular ejection fraction (**vertical axis**) versus preoperative left ventricular end-systolic (END-SYST) volume index (**horizontal axis**) in 56 patients. These two measurements have a significant inverse correlation ($r = -0.80$, $p < 0.001$). All five patients who died suddenly or from heart failure postoperatively showed an end-systolic volume index >200 ml/m^2 and ejection fraction <0.35.

There were significant reductions in the end-diastolic and end-systolic volume indexes in both groups. After operation, the average end-systolic volume index in Group 1 patients showed no notable difference from that in normal subjects. However, Group 2 patients exhibited markedly greater end-diastolic and end-systolic volume indexes than those in normal subjects. The left ventricular mass index also fell in both groups after operation, but did not return to the normal range.

End-systolic wall stress, which was significantly elevated preoperatively in the two patient groups, decreased substantially after surgery; there were no significant differences among the two patient groups and normal subjects (Fig. 5). The ejection fraction, which had decreased remarkably in both patient groups preoperatively, increased significantly. After operation, the average ejection fraction in Group 1 patients (0.62 ± 0.07) was considerably lower than that in normal subjects (0.67 ± 0.06) ($p < 0.05$), but 20 of the 23 patients in Group 1 exhibited normal values. Group 2 patients showed a significantly lower ejection fraction than that in Group 1 patients or normal subjects (Fig. 6); none of those in Group 2 exhibited normalized ejection fraction.

The relation between ejection fraction and end-systolic wall stress was evaluated pre- and postoperatively to evaluate the contractile function of the left ventricle and its change after surgery (Fig. 7). Preoperatively, the two patient groups had a lower ejection fraction with a higher afterload, estimated on the basis of end-systolic wall stress, as compared with normal subjects. There was no marked difference in end-systolic wall stress between the two patient groups. Group 2 patients, however, had a significantly lower ejection fraction than did Group 1 patients, indicating a more depressed contractile function. Postoperatively, the relation between ejection fraction and end-systolic wall stress in many of the Group 1 patients coincided with that of normal subjects. In contrast, patients in Group 2 had a depressed ejection fraction despite a decrease in postoperative end-systolic wall stress to within the normal range, indicating persistently depressed contractile function.

Discussion

Prognostic value of preoperative left ventricular function. Our data support the observation that preoperative left ventricular systolic function is an important determinant of postoperative prognosis, at least in the current operative series of aortic valve replacement for aortic regurgitation (7). Several angiographic variables of left ventricular function were associated with early or late postoperative mor-

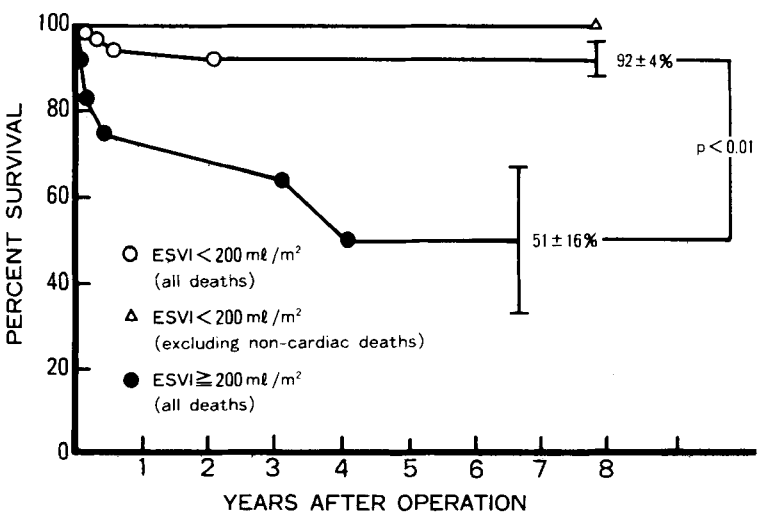


Figure 4. Postoperative survival in 48 patients with preoperative left ventricular end-systolic volume index (ESVI) <200 ml/m^2 versus the 12 patients with a preoperative index >200 ml/m^2.

Table 3. Preoperative Data for 29 Patients Who Underwent Postoperative Studies

Patient No.	Age*/Age† (yr) & Sex	Preoperative CTR (%)	Preoperative NYHA Class	Duration of CHF (mo)	Etiology of AR
Group 1					
1	M40/25	49	I	—	Rheumatic
2	M30/9	48	I	—	Rheumatic
3	M53/36	58	I	—	Rheumatic
4	M29/29	48	II	—	Rheumatic
5	F44/43	53	II	—	Rheumatic
6	F43/42	54	II	—	Rheumatic
7	M26/10	59	II	—	Rheumatic
8	M57/20	61	II‡	—	Rheumatic
9	M48/20	61	II‡	—	Rheumatic
10	M56/23	62	II‡	—	Rheumatic
11	M33/22	56	III	36	Rheumatic
12	M57/49	58	III‡§	5	Rheumatic
13	M33/20	63	III	72	Rheumatic
14	M58/52	73	III§	36	Rheumatic
15	M48/40	50	II	—	IE
16	M47/41	65	II‡	—	IE
17	M43/43	56	III	3	IE
18	M19/17	52	III‡	13	IE
19	M22/12	52	I	—	Bicuspid
20	M38/28	58	III	3	Bicuspid
21	F51/51	58	III‡§	4	Syphilis
22	M41/19	47	II‡	—	Aortitis
23	F48/44	63	IV	48	AAE
Group 2					
24	M55/45	69	II‡	—	Rheumatic
25	M38/20	70	III§	36	Rheumatic
26	M33/13	68	III	54	Rheumatic
27	M48/33	71	IV‡	11	Rheumatic
28	M52/51	64	III‡	9	IE
29	M51/50	74	IV	17	Syphilis

*Age at preoperative catheterization; †age at which heart disease was first noted; ‡angina pectoris; §episode of pulmonary edema. AAE = annuloaortic ectasia; AR = aortic regurgitation; IE = infective endocarditis; other abbreviations as in Table 1.

Table 4. Preoperative and Postoperative Hemodynamic Data for Two Groups of Patients With Aortic Regurgitation

	HR (beats/min)	LVSP (mm Hg)	LVEDP (mm Hg)	CI (liters/min per m^2)	EDVI (ml/m^2)	ESVI (ml/m^2)	LVMI (g/m^2)	EF	ESS (10^3 dynes/cm^2)
Group 1 (n = 23)									
Preoperative	81 ± 19	143 ± 22	15 ± 7	3.00 ± 0.94	216 ± 63	110 ± 45	243 ± 80	0.50 ± 0.08	298 ± 58
Postoperative	78 ± 17	142 ± 24	8 ± 3	3.67 ± 0.91	98 ± 19	37 ± 12	158 ± 40	0.62 ± 0.07	193 ± 45
p Value*	NS	NS	<0.002	<0.005	<0.001	<0.001	<0.001	<0.001	<0.001
Group 2 (n = 6)									
Preoperative	82 ± 20	134 ± 7	19 ± 6	2.06 ± 0.48	379 ± 81	261 ± 52	456 ± 103	0.31 ± 0.03	319 ± 32
Postoperative	77 ± 19	140 ± 23	8 ± 5	2.95 ± 1.12	124 ± 58	74 ± 43	262 ± 103	0.42 ± 0.08	202 ± 49
p Value*	NS	NS	<0.005	<0.05	<0.001	<0.001	<0.001	<0.02	<0.02
Normal subjects (n = 20)									
Mean ± 1 SD	72 ± 13	126 ± 20	9 ± 3	3.85 ± 1.06	82 ± 13	28 ± 7	97 ± 11	0.67 ± 0.06	199 ± 40
p Value†	NS	NS	NS	NS	<0.05	NS	<0.001	<0.05	NS
p Value‡	NS	NS	NS	NS	<0.005	<0.001	<0.001	<0.001	NS

*Compared with preoperative values (paired *t* test); †comparison of normal subjects versus Group 1 patients postoperatively (Neuman-Keuls test); ‡comparison of normal subjects versus Group 2 patients postoperatively (Neuman-Keuls test). Data show mean values ± SD. ESS = end-systolic circumferential wall stress; HR = heart rate; other abbreviations as in Tables 1 and 2.

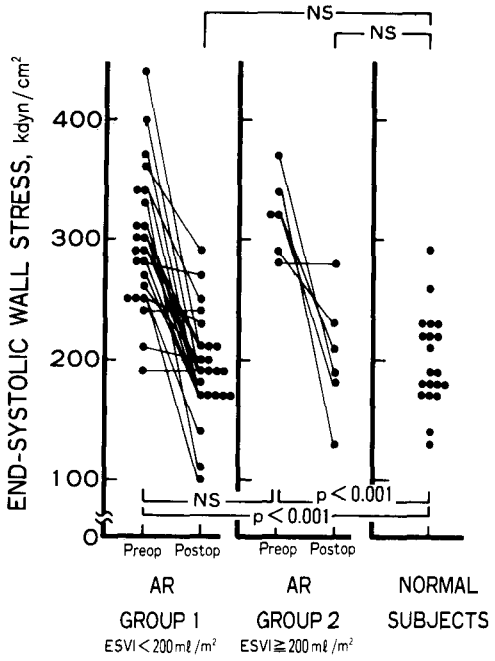
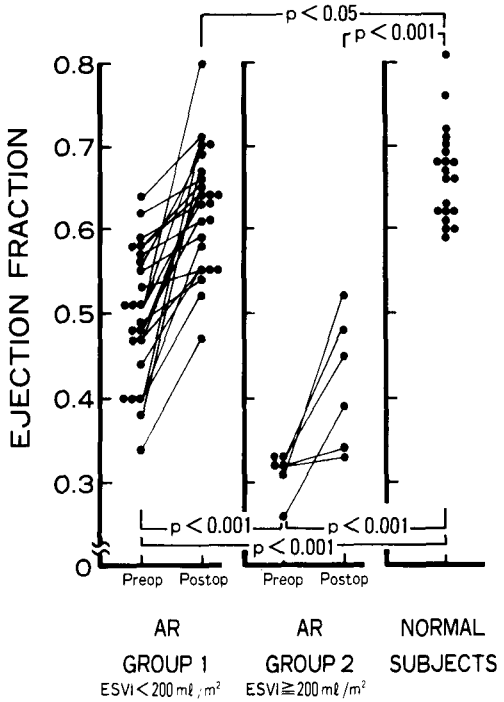


Figure 5. Changes in left ventricular end-systolic wall stress from before (Preop) to after (Postop) aortic valve replacement (AR) for the two patient groups. The values for 20 normal subjects are indicated in the **right** column. ESVI = end-systolic volume index.

Figure 6. Changes in left ventricular ejection fraction from before (Preop) to after (Postop) aortic valve replacement (AR). The values for 20 normal subjects are indicated in the **right** column. Twenty of the 23 patients in Group 1 showed a normal ejection fraction (within 2 SD of normal) after operation. None of the six patients in Group 2 showed normalization of ejection fraction. ESVI = end-systolic volume index.



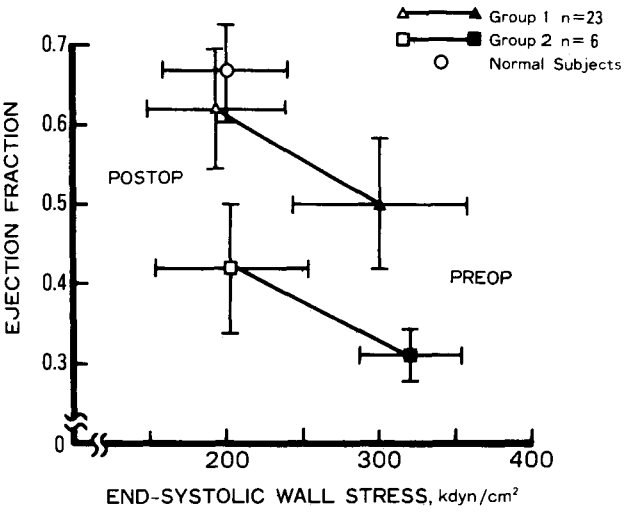


Figure 7. Relation between ejection fraction and end-systolic wall stress and its change from before (PREOP) to after (POSTOP) for the two patient groups. The relation for 20 normal subjects is also indicated. It appears that the increase in ejection fraction is secondary to a reduction in end-systolic stress and not to an intrinsic change in myocardial function. Data are shown as mean ± 1 SD.

tality and persistent left ventricular dysfunction. Among angiographic variables examined, the preoperative end-systolic volume index proved to be closely associated with an unfavorable postoperative outcome. Our study, moreover, provides additional information regarding the minimal level of preoperative left ventricular function necessary for satisfactory long-term survival and hemodynamic results postoperatively.

The five patients who had a cardiac death in our series, when compared with late survivors, had a very large, poorly contracting left ventricle before surgery; all five patients had an end-systolic volume index >200 ml/m^2 and an ejection fraction <0.35. These five patients constituted 42% of the 12 patients in our series who had a preoperative end-systolic volume index >200 ml/m^2. In contrast, none of the 48 patients with a lower end-systolic volume index died of cardiac-related causes.

Several studies have demonstrated the importance of preoperative ejection fraction as a predictor of postoperative prognosis in aortic regurgitation (1,3,5). Borow et al. (18), however, reported that the preoperative end-systolic volume index was a more reliable predictor of postoperative survival and functional results than was the preoperative ejection fraction. Their data were consistent with the findings of Grossman et al. (19) that the end-systolic volume alone can be a useful measure of myocardial function. The findings of Borow et al. were also supported by those of Henry et al. (4) who, using echocardiographic studies, suggested that if the preoperative left ventricular end-systolic dimension measured by M-mode echocardiography was >55 mm, a high percentage of patients developed late heart failure or

died after operation. In contrast, Greves et al. (5) reported no difference in late mortality between patients with and without a preoperative end-systolic volume index >110 ml/m^2. However, the preoperative end-systolic volume index and ejection fraction values are closely correlated (Fig. 3). We believe that these two variables have similar prognostic values for aortic regurgitation.

Postoperative ventricular performance and wall stress. Although the patients in our series with aortic regurgitation and severely impaired left ventricular function survived surgery (no patient died at operation), they are considered at high risk of dying from myocardial failure late after operation. We therefore examined left ventricular contractile function in these patients after aortic valve replacement.

Our most important finding was that end-systolic wall stress, which was elevated preoperatively, returned to the normal range, allowing an increase in ejection fraction for both patient groups after aortic valve replacement (correction of afterload mismatch). However, despite this favorable change in loading conditions, the ejection fraction in Group 2 patients (preoperative end-systolic volume index >200 ml/m^2) failed to return to normal, indicating that associated irreversible myocardial dysfunction was primarily responsible for the reduced ejection fraction in these patients.

Numerous studies (7, 20–26) have indicated that impaired preoperative left ventricular function may improve and often become normalized after valve replacement. Clark et al. (25) found in 17 patients with symptomatic aortic regurgitation and impaired left ventricular function (ejection fraction <0.50) that postoperative left ventricular systolic function improved in 50% of the patients. Schwarz et al. (24) showed that impaired preoperative left ventricular function (all patients displayed a preoperative ejection fraction >0.35) became completely normalized after valve replacement with a Björk-Shiley valve if valve function was good. These studies, however, have not defined what level of depressed left ventricular function or what degree of dilation of the left ventricle would indicate irreversible myocardial dysfunction. Borow et al. (18) observed that patients with a preoperative end-systolic volume index >90 ml/m^2, measured by contrast angiography, exhibited a reduced fractional shortening on echocardiography and tended to remain in functional class III or to die after surgery. However, in our current study, many of the patients with a preoperative end-systolic volume index >100 ml/m^2 showed lessening of symptoms and improved left ventricular function postoperatively. We speculate that, owing to improved myocardial preservation techniques, patients currently undergoing aortic valve replacement will display better restoration of left ventricular function than did those in earlier studies.

Limitations of the method. Our wall stress measurements have several limitations. First, our pressures were recorded just before but not simultaneously with left ventriculography. However, it is unlikely that systolic pressures changed in the interval between pressure recording and ventriculography. In postoperative patients in whom aortic pressure recording was performed just before and simultaneously with angiography, we did not see any significant changes either in systolic pressure or in heart rate. Second, there may be small errors in our stress measurements attributable to the use of a fluid-filled catheter. However, because the difference in peak systolic pressure measured with a well flushed fluid-filled catheter and a micromanometer-tipped catheter is small, no serious errors are thought to be produced by this method. Third, peak systolic pressure was substituted for end-systolic pressure. As discussed by Grossman et al. (19), however, peak systolic pressure usually occurs close to the point of minimal left ventricular volume (27). Other studies (28) have indicated that peak and end-systolic pressures are nearly equal in magnitude although they occur at different times. Reichek et al. (29) demonstrated a close correlation between left ventricular peak systolic and end-systolic pressures ($r = 0.97$). Fourth, some of the patients had atrial fibrillation, in which beat to beat differences in stress and contractility are found. To minimize error in stress calculations in patients with atrial fibrillation, pressure and volume were carefully matched by the RR interval obtained from electrocardiographic (ECG) recordings made before and during angiography. Finally, our stress values are estimates of true "end-systolic" stress and are used to analyze pre- and postoperative changes in afterload and to compare the two patient groups with normal subjects.

Clinical implications. Of importance is the fact that patients with aortic regurgitation and a preoperative end-systolic volume index >200 ml/m^2 are at high risk of incurring irreversible myocardial dysfunction and of dying postoperatively either suddenly or from congestive heart failure. These patients have unusually serious cardiac enlargement compared with that of patients reported on by other investigators. These patients in our series underwent operation too late in the course of their disease; some of them had refused an operation for years because of few subjective symptoms, and surgical intervention was delayed too long as a result. The other patients were referred to our institution for surgery after severe clinical and hemodynamic deterioration had occurred.

The present study comprised a rather small number of patients, but included all those at our institution reliably known to have isolated, chronic aortic regurgitation, as well as a wide spectrum of patients in relation to both severity of cardiac symptoms and degree of left ventricular function. We assume that the results of our current operative series also correspond to those of other institutions.

Conclusion. Our data indicate that in current surgery for aortic regurgitation, the minimal preoperative left ventricular function necessary for satisfactory postoperative survival and functional results may be at a lower level (that

is, end-systolic volume index 200 ml/m^2 and ejection fraction 0.35) than has been suggested in previous studies (4,6,18,24).

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